b.) Remarks

Claims 1 and 8 have been amended in order to recite the present invention with the specificity required by statute. Additionally, claims 4 and 11 are amended for better idiomatic format.

The subject matter of the amendment may be found in the specification as filed, *inter alia*, in the paragraph bridging page 7 and page 8. Accordingly, no new matter has been added.

Claims 1-5 and 8-12 remain rejected as being obvious over the art of record namely Suzuki, Trenkwalder and Endente, for the reasons of record. Specifically, the Examiner states she has considered Dr. Kanda's Declaration but

> "Parkinson's disease increases the risk of RLS or nocturnal myoclonus or vice versa. As discussed [above], the Examiner considers RLS and nocturnal myoclonus to be symptoms in Parkinson's disease patients. As taught by Trenkwalder, it would seem that the 60-90% of patients that complain about said secondary mechanisms would benefit from a treatment agent that [is] useful in treating such a disease. Thus, it would seem obvious that by administering PD [patients] with Applicant's compound, that one would also be able to treat the symptoms thereof including restless leg syndrome and/or nocturnal myoclonus in said overlapping population of patients" (Office Action, page 3, lines 5-12, emphases added).

Initially, Applicants respectfully wish to point out the Examiner's rejection explicitly relies on the apparent inherency of Parkinson's disease patients as presenting RLS or noctural myoclonus[1]. However, the rejection then is without bases in law since it is not permitted to rely on inherency for obviousness. *In re Spormann*, 363 F.2d 444 (CCPA 1966); *In re Dillon*, 919 F.2d 688 (Fed. Cir. 1990) (en banc), cert. denied sub nom. *Dillon v. Manbeck*, 500 US 904 (1991).

Nonetheless, solely in order to reduce the issues and expedite prosecution, Applicants have above amended their claims so as to exclude patients suffering from Parkinson's disease. There is clearly no reason of record for administering an adenosine A_{2A} receptor antagonist (which is well-known as an anti Parkinsonian agent) to treat patients not suffering from Parkinson's disease. Accordingly, the rejection of claims 1-5 and 8-12 is mooted.

In view of the above amendments and remarks, Applicants submit that all of the Examiner's concerns are now overcome and the claims are now in allowable condition. Accordingly, reconsideration and allowance of this application is earnestly solicited.

Claims 1-5 and 8-12 remain presented for continued prosecution.

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[1] Also, to complete the record, Applicants note the Examiner points out that in the strain of mouse employed in the Parkinson's disease model of Suzuki, striatal dopamine is decreased (and locomotor activity is depressed), and concludes it is evident that Suzuki's xanthine derivatives play a role in the dopaminergic system. However, although Suzuki's xanthine derivatives successfully regain locomotor activity, Suzuki is silent about the effect on any striatal dopamine level. In that regard, Shiozaki et al., *Psychopharmacology*, Vol. 147 (1999) 90-5 (cited in the July 22, 2009 Information Disclosure Statement) reports that adenosine A_{2A} receptor antagonist exerts the efficacy completely through the different target molecule and mechanism of action from dopaminomimetic drugs such as L-DOPA. See second paragraph to last paragraph of right column of p. 94.

Applicants' undersigned attorney may be reached in our New York office by telephone at (212) 218-2100. All correspondence should continue to be directed to our below listed address.

Respectfully submitted,

/Lawrence S. Perry/
Lawrence S. Perry
Attorney for Applicants
Registration No. 31,865

FITZPATRICK, CELLA, HARPER & SCINTO
1290 Avenue of the Americas
New York, New York 10104-3800
Facsimile: (212) 218-2200

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